Original Article

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Biochemistry Section

Effect of Dialysis on Iron Profile and Haematological Parameters in Patients with Chronic Kidney Disease: A Retrospective Observational Study

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ABSTRACT

Introduction: Iron is an essential mineral to trigger erythropoiesis in response to Erythropoietin (EPO) from kidneys. In Chronic Kidney Disease (CKD), dialysis is a treatment option when the kidneys fail to filter out the toxins from blood which may cause absolute or relative iron deficiency causing anaemia.

Aim: To elucidate significance of iron profile in dialysis cases for management of anaemia post-dialysis in male and female patients with CKD.

Materials and Methods: The present retrospective observational study was conducted from September 2023 to November 2023 at Sri Devaraj Urs Medical College and Hospital, Kolar, Karnataka, India. A total of 84 subjects including male and female patients undergoing dialysis with or without diabetes mellitus, CKD cases and Haemodialysis cases were included in the study. Latest and recent values of iron profile and Complete Blood Count (CBC) parameters of patients were collected from biochemistry and haematology sections for the study, the statistical analysis was performed with Statistical Package for Social Sciences (SPSS) version 25 (IBM).

Results: The Transferrin Saturation (TSAT) was comparable between males (median 22.74%, IQR: 15.34-31%) and females (median 24.3%, IQR: 15.7-30.4%) (p=0.47). Non-diabetic patients showed higher TSAT (median 22.74%, IQR: 15-32%) compared to diabetics (median 24.3%, IQR: 15.7-30.4%). The Unsaturated Iron Binding Capacity (UIBC) was 160.1±60.2 µg/dL in males and 164.6±38.61 µg/dL in females (p=0.73), being higher in non-diabetics (165±515.43 µg/dL) than diabetics (156.4±58.3 µg/dL) (p=0.5), demonstrating the inverse relationship with serum iron as per iron physiology.

Conclusion: Iron profile assessment including calculated parameters (TSAT and UIBC) is essential for precision management of anaemia in dialysis patients. There was no significant gender-based difference but there was notable variation between diabetic and non-diabetic CKD cases.

Keywords: Erythropoiesis, Renal disease, Transferrin saturation, Unsaturated iron binding capacity

INTRODUCTION

Dialysis is a treatment option when the kidneys fail to filter out the toxins from blood. There are two types of dialysis; Haemodialysis and peritoneal dialysis [1]. Dialysis shall be performed in cases with Acute Kidney Injury (AKI) and/or CKD [2]. There are five stages in CKD, stages 4 and 5 cases are recommended to undergo dialysis [2].

Renal tissues produce EPO; a hormone responsible to initiate erythropoiesis from the bone marrow [3]. During renal insufficiency the hormone secretion is decreased there by decrease in blood cells. During dialysis absolute iron deficiency shall be observed due to blood loss or impaired iron absorption or restricted diet or uraemia [2]. The anaemia worsens from stages 1 to 5 leading to increased morbidity and mortality [2].

Iron is an essential mineral to trigger erythropoiesis in response to EPO from kidneys [4]. Iron is recycled from senescent RBC and a very little is acquired through the diet which is regulated by Hepcidin [2]. Therefore, in CKD there may be absolute or relative iron deficiency causing anaemia. There are various other metabolic functions of iron such as activator and carrier which when depleted leads to several pathological conditions [3]. The pathophysiological CKD-anaemia mechanism is still not clearly elucidated.

Diagnosis of pathophysiological CKD-anaemia is by biochemical iron profile test and haematological CBC. Iron Deficiency Anaemia (IDA) is assessed by Haemoglobin (Hb %) and haematocrit, reticulocyte count, Mean Corpuscular Hb (MCH), and Mean Corpuscular Volume (MCV). To classify whether anaemia is an absolute or relative deficiency of iron, advanced biochemical parameters such as Total Iron Binding Capacity (TIBC), ferritin and transferrin are estimated [5]. In clinical practice and for management of anaemia in dialysis dependent CKD cases, TSAT is calculated [6].

The novelty of this study lies in the comprehensive evaluation of calculated iron parameters (TSAT and UIBC) in conjunction with directly measured iron profile parameters in dialysis-dependent CKD patients from this regional population. While iron profiling is routinely performed, the systematic calculation and analysis of TSAT and UIBC across different metabolic subgroups (diabetic vs non-diabetic) remains underexplored in clinical practice, particularly in this geographic area. These calculated parameters provide critical insights into iron storage and transport mechanisms that guide precision iron supplementation therapy, yet are often overlooked in routine clinical assessment of dialysis patients. Since intravenous administration of iron without transferrin and TSAT values shall be fatal, renal association and National Institute for Healthcare and Excellence guidelines recommend transferrin estimation and TSAT calculation [6,7]. Despite the well-established relationship between iron deficiency and anaemia in CKD patients, there is limited data on calculated iron parameters (TSAT and UIBC) in dialysis-dependent patients from the study region, necessitating this investigation to guide optimal iron supplementation strategies in clinical practice.

The present study aimed to elucidate significance of iron profile in dialysis case for management of anaemia post dialysis in male and female CKD patients and to correlate biochemical and haematological parameters and to calculate TSAT and UIBC.

MATERIALS AND METHODS

The present retrospective observational study was conducted from September 2023 to November 2023 at Sri Devaraj Urs Medical College and Hospital, a Tertiary Care Hospital in Kolar, Karnataka. In the present study, data were collected between September 2023 to November 2023 and data were analysed between December 2023 to January 2024. The study was initiated after obtaining the ethical clearance from the Institutional Ethics Committee (DMC/KLR/IEC/714/2022-23). The data were collected after obtaining verbal consent from the study.

Inclusion criteria: All the male and female undergoing dialysis with or without diabetes mellitus, with CKD were included.

Exclusion criteria: Subjects with AKI, cancer patients, age <25 years and > 70 years, drug induced renal damage were excluded from the study.

Sample size calculation: Mean difference in haemoglobin levels between male and female haemodialysis patients was calculated with 80% power and 95% confidence interval. Based on literature data [8], the sample size of 84 CKD cases was determined.

Formula for comparing means: $N = \{2Sp^2 (Z1\text{-}\alpha/2 + Z1\text{-}\beta)^2\}/\mu d^2$

Where: $Sp^2 = (S1^2 + S2^2)/2$

S1 (Standard Deviation in males): 2.4 g/dL for haemoglobin

S2 (Standard Deviation in females): 2.5 g/dL for haemoglobin

μd (Mean difference between groups): 0.8 g/dL

Sp^2 (Pooled variance): $\{(2.4)^2 + (2.5)^2\}/2 = 6.005$

α (Level of significance): 0.05 (5%)

1-β (Power): 0.80 (80%)

Z1-α/2 (at 95% confidence interval): 1.96

Z1-β (at 80% power): 0.84

Groups compared: Male and female haemodialysis patients

Calculated sample size per group: 42 (Total: 84 CKD cases)

Haemoglobin was used as the primary parameter for sample size calculation as it is the most clinically relevant indicator of anaemia management in dialysis patients.

[Table/Fig-1] outlines the methodological framework for correlation analysis in this study [2,6]. Iron binding parameters (TIBC and UIBC), renal function markers (blood urea and creatinine) and inflammatory markers (total leucocyte count) were assessed in present study. All parameters were measured using standardised automated methods on validated analysers (Vitros 5.1 FS and Sysmex systems), ensuring analytical precision.

Study Procedure

The latest and recent values of iron profile and CBC parameters of patients were collected from biochemistry and haematology sections of Central Diagnostic Laboratory Services (CDLS) of a Tertiary Rural Care Hospital in Southern part of Karnataka, India. The data was compiled and entered in excel workbooks without names of the patients until the desired number of cases are reached.

Parameter	Method of estimation	Reference range	Reference
Iron binding parameters			
Total Iron Binding Capacity (TIBC)	Direct measurement using excess iron binding followed by colorimetric detection (Vitros 5.1 FS analyser)	250-450 µg/dL	[6]
Unsaturated Iron Binding Capacity (UIBC)	Calculated: TIBC - serum iron	111-343 µg/dL	[6]
Renal function markers			
Blood urea	Enzymatic urease method (Vitros 5.1 FS analyser)	15-40 mg/dL (2.5-6.7 mmol/L)	[2]
Serum creatinine	Enzymatic creatinine assay - creatininase-creatinase-sarcosine oxidase-peroxidase sequence (Vitros 5.1 FS analyser)	Males: 0.7-1.3 mg/dL; Females: 0.6-1.1 mg/dL	[2]
Inflammatory marker			
Total Leucocyte Count (TLC)	Electrical impedance, hydrodynamic focusing, and flow cytometry with semiconductor laser (Sysmex 5-part hematology analyser)	4,000-11,000/µL	TIBC, UIBC (within same gender) [2]

[Table/Fig-1]: Correlation parameters, methods of estimation, reference ranges, and analytical approach [2,6].
TIBC: Total iron binding capacity; UIBC: Unsaturated iron binding capacity; TLC: Total leucocyte count

[Table/Fig-2] summarises the laboratory methods, instruments, and reference ranges used to assess biochemical, iron, and haematological parameters. Standardised assays on the Vitros 5.1 FS and **Sysmex 5-part analysers** ensured precision and consistency. Calculated indices- TSAT and UIBC were derived using standard formulas to evaluate iron availability and transport.

Parameters	Method of estimation	Cut-off range	Reference
Biochemical parameters			
Serum urea	Enzymatic urease method (Vitros 5.1 FS analyser)	15-40 mg/dL (2.5-6.7 mmol/L)	[2]
Serum creatinine	Enzymatic creatinine assay-creatininase–creatinase-sarcosine oxidase-peroxidase sequence (Vitros 5.1 FS analyser)	Males: 0.7-1.3 mg/dL; Females: 0.6-1.1 mg/dL	[2]
Iron profile parameters			
Serum iron	Ferrozine-based colorimetric method (Vitros 5.1 FS analyser)	Males: 65-175 µg/dL; Females: 50-170 µg/dL	[6,7]
Total Iron Binding Capacity (TIBC)	Direct measurement using excess iron binding followed by colorimetric detection (Vitros 5.1 FS analyser)	250-450 µg/dL	[6]
Transferrin	Immunoturbidimetric method (Vitros 5.1 FS analyser)	200-360 mg/dL	[6]
Serum Ferritin	Chemiluminescence immunoassay - CLIA (Vitros eCI system)	Males: 30-400 ng/mL; Females: 13-150 ng/mL	[2,6]
Calculated iron parameters			
Transferrin Saturation (TSAT)	Calculated: (serum iron ÷ TIBC) × 100	>20% (optimal: 20-50%)	[6]
Unsaturated Iron Binding Capacity (UIBC)	Calculated: TIBC - serum iron	111-343 µg/dL	[6]
Haematological parameters			
Complete Blood Count (CBC)	Electrical impedance, hydrodynamic focusing, and flow cytometry with semiconductor laser (Sysmex 5-part hematology analyser)	See individual parameters below	[2]
Haemoglobin (Hb)	CBC - Sysmex 5-part analyser	Males: 13.5-17.5 g/dL; Females: 12-16 g/dL	[2,3]
Total Leukocyte Count (TLC)	CBC - Sysmex 5-part analyser	4,000-11,000/µL	[2]
Platelet Count	CBC - Sysmex 5-part analyser	150,000-450,000/µL	[2]
Red Blood Cell Count (RBC)	CBC - Sysmex 5-part analyser	Males: 4.7-6.1 $\times 10^6$/µL; Females: 4.2-5.4 $\times 10^6$/µL	[2,3]

[Table/Fig-2]: Laboratory methods, reference ranges, and citations [2,3,6,7].
CBC: Complete blood count; TIBC: Total iron binding capacity; TSAT: Transferrin saturation; UIBC: Unsaturated iron binding capacity; CLIA: Chemiluminescence immunoassay

STATISTICAL ANALYSIS

The SPSS version 25 (IBM corp.) was used to perform statistics. All the variables which are normally distributed (Parametric) are represented as mean±SD. Analysis of Variance (ANOVA) was applied to find probability- value (p-value) across groups, Pearson's correlation (r) to find the trend between two variables (either positively correlated or negatively correlated). Non-parametric variables are represented as median (25^{th}- 75^{th} percentile), Kruskal- Walli's to derive the p-value across groups. Spearmann's Rho (ρ) correlation was applied to find whether the non-parameters variables are negatively or positively correlated Normality of distribution of variables will be assessed by Kolmogorov- Smirnov test. The p-value <0.05 was considered statistically significant.

RESULTS

In the present study, the mean age of male participants (69±10.05 years) was comparable to female participants (71±12.11 years) with no statistical significance (p=0.56). Similarly, both systolic and diastolic blood pressures showed no significant gender-based differences, indicating homogeneity in baseline cardiovascular parameters between groups [Table/Fig-3].

Parameters	Male (54)	Female (30)	p-value
Age (in years)	69±10.05	71±12.11	0.56
SBP (mmHg)	132±10.6	132±8.15	0.97
DBP (mmHg)	90.7±6.91	88.1±5.7	0.094

[Table/Fig-3]: Demographic details represented as mean±Standard deviation.
SBP: Systolic blood pressure, DBP: Diastolic blood pressure

As shown in [Table/Fig-4], non-parametric variables including random blood sugar, blood urea, creatinine, serum iron, TSAT, and total leucocyte count revealed no statistically significant differences between male and female dialysis patients (all p>0.05). The median TSAT in both genders remained below the optimal threshold of >50%, indicating inadequate iron availability for erythropoiesis in the study population.

Parameters	Male (54)	Female (30)	p-value
RBS (140- 200mg/dL)	110.35 (91.5-156.5)	89 (73-151)	0.76
Blood urea (15- 35mg/dL)	100 (78-137)	98(70-148)	0.5
Creatinine (0.6- 1.4mg/dL)	6.6 (4.6-10.83)	4.8 (3.6-8.6)	0.11
Serum iron (26-170 µg/dL in women 76-198 µg/dL in men)	52 (33.8-65.3)	41.5 (34-69.3)	0.7
TSAT % (>50%)	22.74 (15.34-31)	24.3 (15.7-30.4)	0.47
TLC (4.5-11.0 × 10^9/L)	11.53 (7.72-20.61)	14 (8.2- 18.8)	0.95

[Table/Fig-4]: Gender based comparison of median (25th - 75th percentile).
RBS: Random blood sugar; TSAT: Transferrin saturation; TLC: Total leucocyte count

In the present study, total iron binding capacity (males: 214.9±5.8 µg/dL vs females: 217.6±45.7 µg/dL), transferrin, UIBC, haemoglobin, and RBC counts showed no significant gender-based variations (all p >0.05). Both groups demonstrated haemoglobin levels below normal reference ranges, confirming the presence of anaemia in dialysis-dependent CKD patients irrespective of gender [Table/Fig-5].

Parameters	Male (54)	Female (30)	p-value
TIBC (262-474 µg/dL)	214.9±5.8	217.6±45.7	0.8
Transferrin (204-360 mg/dL)	166±51.1	170±46.2	0.8
UIBC (111 mcg/dL to 343 mcg/dL)	160.1±60.2	164.6±38.61	0.73
Hb (13.7-17.5 g/dL in male 11.5 11.5-15 g/dL)	8.7±2.4	8.3±2.5	0.58
RBC (4.0 to 5.9 x 10^{12}/L. women – 3.8 to 5.2 x 10^{12}/L.)	3.3±0.8	3.2±0.7	0.74

[Table/Fig-5]: Gender based mean± standard deviation of parametric variables.
TIBC: Total iron binding capacity; UIBC: Unsaturated iron binding capacity; Hb: Haemoglobin; RBC: Red blood corpuscles

Correlation analysis was performed to understand the relationship between iron metabolism parameters (TIBC and UIBC) and renal function markers (blood urea and creatinine) as well as inflammatory markers (TLC) within each gender group. This gender-specific correlation analysis was conducted to identify whether iron binding capacity patterns differ between males and females in response to varying degrees of renal dysfunction and inflammation

Comprehensive correlation analysis between iron profile and clinical parameters has been shown in [Table/Fig-6a-c].

[Table/Fig-6a] reveals significant correlations between iron parameters and renal function in males. TIBC and UIBC showed positive correlations with blood urea (r=0.48, p=0.02; r=0.43, p=0.03) and negative correlations with creatinine (r=-0.52, p=0.01; r=-0.46, p=0.02), indicating that deteriorating renal function impairs iron binding capacity. Serum iron and TSAT correlated negatively with blood urea (r=-0.38, p=0.042; r=-0.41, p=0.035), confirming that uraemia reduces iron availability for erythropoiesis. No significant correlations were observed with RBS, suggesting glycaemic status has minimal direct impact on iron metabolism in male dialysis patients.

Iron parameters (Males)	Blood urea (males)	Creatinine (Males)	RBS (Males)
Serum iron (µg/dL)	r=-0.38, p=0.042	r=0.35, p=0.058	r=-0.21, p=0.13
TIBC (µg/dL)	r=0.48, p=0.02	r=-0.52, p=0.01	r=0.19, p=0.17
UIBC (µg/dL)	r=0.43, p=0.03	r=-0.46, p=0.02	r=0.23, p=0.09
TSAT (%)	r=-0.41, p=0.035	r=0.39, p=0.041	r=-0.18, p=0.19
Transferrin (mg/dL)	r=0.44, p=0.028	r=-0.49, p=0.015	r=0.17, p=0.21

[Table/Fig-6a]: Correlation between iron parameters and renal function markers in males.
TIBC: Total iron binding capacity; UIBC: Unsaturated iron binding capacity; TSAT: Transferrin saturation; RBC: Red blood cell; TLC: Total leucocyte count; r: Pearson's/Spearman's correlation coefficient

[Table/Fig-6b] demonstrates similar correlation patterns in females as observed in males, confirming gender-independent relationships between iron metabolism and renal function. TIBC and UIBC positively correlated with blood urea (r=0.49, p=0.015; r=0.44, p=0.025) and negatively with creatinine (r=-0.50, p=0.012; r=-0.47, p=0.018). TSAT showed inverse correlation with blood urea (r=-0.39, p=0.038), while serum iron's negative correlation with blood urea approached significance (r=-0.36, p=0.051). These findings reinforce that progressive renal impairment universally disrupts iron homeostasis regardless of gender.

Iron parameters (females)	Blood urea (females)	Creatinine (females)	RBS (females)
Serum iron (µg/dL)	r=-0.36, p=0.051	r=0.33, p=0.072	r=-0.19, p=0.31
TIBC (µg/dL)	r=0.49, p=0.015	r=-0.50, p=0.012	r=0.21, p=0.26
UIBC (µg/dL)	r=0.44, p=0.025	r=-0.47, p=0.018	r=0.24, p=0.20
TSAT (%)	r=-0.39, p=0.038	r=0.37, p=0.045	r=-0.16, p=0.40
Transferrin (mg/dL)	r=0.46, p=0.020	r=-0.48, p=0.016	r=0.18, p=0.34

[Table/Fig-6b]: Correlation between iron parameters and renal function markers in females.
TIBC: Total iron binding capacity; UIBC: Unsaturated iron binding capacity; TSAT: Transferrin saturation; RBC: Red blood cell; TLC: Total leucocyte count; r: Pearson's/Spearman's correlation coefficient

[Table/Fig-6C] illustrates the functional impact of iron parameters on erythropoiesis. In both genders, serum iron and TSAT demonstrated strong positive correlations with haemoglobin (males: r=0.52, p=0.009; r=0.58, p=0.003; females: r=0.49, p=0.016; r=0.55, p=0.006) and RBC count, confirming their critical role in red blood cell production. Transferrin showed positive correlation with TLC in both males (r=0.38, p=0.045) and females (r=0.40, p=0.035), suggesting potential inflammatory influences on iron transport proteins. These correlations validate that adequate iron availability, reflected by higher TSAT values, directly translates to improved haemoglobin synthesis and erythropoiesis in dialysis patients.

Iron Parameters	Haemoglobin	RBC Count	TLC
In males:			
Serum iron	r=0.52, p=0.009	r=0.48, p=0.015	r=-0.28, p=0.08
TSAT	r=0.58, p=0.003	r=0.51, p=0.011	r=-0.31, p=0.06
Transferrin	r=-0.35, p=0.055	r=-0.32, p=0.071	r=0.38, p=0.045
In females:			
Serum iron	r=0.49, p=0.016	r=0.45, p=0.023	r=-0.26, p=0.16
TSAT	r=0.55, p=0.006	r=0.48, p=0.017	r=-0.29, p=0.12
Transferrin	r=-0.33, p=0.069	r=-0.30, p=0.10	r=0.40, p=0.035

[Table/Fig-6c]: Correlation between iron parameters and haematological markers.
TIBC: Total iron binding capacity; UIBC: Unsaturated iron binding capacity; TSAT: Transferrin saturation; RBC: Red blood cell; TLC: Total leucocyte count; r: Pearson's/Spearman's correlation coefficient

As depicted in [Table/Fig-7], diabetic patients showed significantly higher random blood sugar levels (median 107 mg/dL, IQR: 73-161) compared to non-diabetics (median 97 mg/dL, IQR: 56.3-116.3) (p=0.016). Interestingly, non-diabetic patients demonstrated significantly elevated blood urea (median 118 mg/dL, IQR: 85.5-166) compared to diabetics (median 98 mg/dL, IQR: 70-148) (p=0.023), indicating potentially more severe renal impairment in the non-diabetic CKD group. TSAT and other iron parameters showed no significant differences between diabetic status groups.

Parameters	Non- Diabetic Group (39)	Diabetic Group (45]	p-value
RBS mg/dL	97 (56.3-116.3)	107 (73-161)	0.016*
Blood Urea mg/dL	118 (85.5-166)	98 (70-148)	0.023*
Serum Creatinine mg/dL	6.6 (4.2-10.7)	4.8 (3.6-8.6)	0.216
Serum iron mg/dL	51 (28.8-66)	41.5 (34-69.3)	0.6
TSAT %	22.74 (15-32)	24.3 (15.7-30.4)	0.95
TLC x10^9/L	12 (8-20.8)	14 (8.2-18.8)	0.32

[Table/Fig-7]: Comparison between diabetic and non- diabetic subjects represented median (25th- 75th percentile).
RBS: Random blood sugar, TSAT: Total saturation, TLC: Total leucocyte count

[Table/Fig-8] compares iron profile and haematological parameters between diabetic and non-diabetic groups. Although non-diabetic patients showed numerically higher TIBC (220±48.5 µg/dL), transferrin (170.9±46.3 mg/dL), and UIBC (165±515.43 µg/dL) compared to diabetics, these differences did not reach statistical significance (all p>0.05). Both groups exhibited similar degrees of anaemia with haemoglobin levels around 8.4-8.8 g/dL, emphasising the universal impact of CKD on erythropoiesis regardless of diabetic status.

Parameters	Non-diabetic group (39)	Diabetic group (45)	p-value
TIBC µg/dL	220±48.5	209±52.3	0.35
Transferrin mg/dL	170.9±46.3	163.2±53.9	0.5
UIBC µg/dL	165±515.43	156.4±58.3	0.5
Haemoglobin %	8.8±2.7	8.4 ±2.1	0.43
RBC x10^{12}/L	3.6±0.9	3.2±0.56	0.89

[Table/Fig-8]: Comparison between diabetic and non-diabetic subjects represented as mean±SD.
TIBC: Total iron binding capacity; UIBC: Unsaturated iron binding capacity; RBC: Red blood corpuscles

DISCUSSION

The demographic analysis revealed that blood pressure control in CKD patients undergoing dialysis was not influenced by gender, with both groups showing comparable systolic (132±10.6 mmHg in males vs 132±8.15 mmHg in females) and diastolic pressures. This finding aligns with the study by de Hauteclocque A et al., who reported similar cardiovascular parameters across genders in dialysis populations [9].

The biochemical and haematological parameters comparison between males and females revealed statistically insignificant variations across all measured parameters. This contrasts with studies by Yu MK et al., and Ciarambino T et al., which reported gender-specific differences in renal function decline [10,11]. However, the present findings support the hypothesis that in advanced CKD requiring dialysis, the disease severity may override gender-specific physiological differences, resulting in comparable metabolic derangements. The sample size disparity (54 males vs 30 females) in the present study reflected the clinical reality of higher male representation in dialysis units during the study period, consistent with observations by de Hauteclocque A et al., [9].

The correlation analysis demonstrated significant relationships between iron binding capacity and renal function markers. Serum creatinine showed inverse correlation with both TIBC (r=-0.51, p=0.008) and UIBC (r=-0.48, p=0.013), indicating that deteriorating renal function directly impairs iron binding capacity. This finding validates the observations by Jurkovitz CT et al., who established that elevated serum creatinine is associated with decreased iron availability [12]. Furthermore, the positive correlation between TLC and iron binding capacity (r=0.424, p=0.031; r=0.52, p=0.07) corroborates the work of AlRajeh L et al., in populations with low iron concentrations, suggesting that inflammatory processes reflected by leucocyte counts may influence iron metabolism in CKD patients [13].

The calculated parameters TSAT and UIBC provided valuable insights into iron storage and transport mechanisms. TSAT was comparable between genders (males: 22.74% vs females: 24.3%, p=0.47) but notably higher in non-diabetics (median 22.74%) compared to diabetics (median 24.3%) at the 75th percentile. Both groups demonstrated TSAT values below the optimal threshold of >50%, indicating functional iron deficiency. These findings align with Bahrainwala J and Berns JS who emphasised the utility of TSAT in diagnosing iron deficiency in CKD patients and reported similar gender-independent patterns [6]. The UIBC values were elevated in females (164.6±38.61 µg/dL) compared to males (160.1±60.2 µg/dL), reflecting increased transferrin concentrations in females. This inverse relationship between transferrin levels and iron binding capacity is consistent with established iron physiology, as documented by Kuragano T et al., who demonstrated that UIBC represents the unutilised iron-binding capacity of transferrin [14].

The comparison between diabetic and non-diabetic groups yielded significant findings. While diabetic patients showed predictably higher blood glucose levels (median 107 mg/dL vs 97 mg/dL, p=0.016), non-diabetic patients demonstrated significantly higher blood urea (median 118 mg/dL vs 98 mg/dL, p=0.023), suggesting more advanced uraemia. This unexpected finding indicates that non-diabetic CKD patients in our cohort, primarily with hypertensive nephropathy, exhibited more severe renal impairment than their diabetic counterparts. This contrasts with Fujii M et al., who reported accelerated renal decline in diabetic patients in the Japanese population [15]. The discrepancy may be attributed to better glycaemic control and use of renoprotective diabetic medications in our diabetic cohort, whereas hypertensive patients may have had suboptimal blood pressure management. The iron binding capacities were higher in non-diabetics than diabetics, which AlQarni AM et al., explained as a consequence of glycaemic status affecting iron metabolism, demonstrating that HbA1c levels influence iron replacement therapy effectiveness [16].

The present study's findings validate the importance of comprehensive iron profiling in dialysis patients across different demographic and metabolic subgroups, supporting the recommendations by Mikhail A et al., and Kidney Disease Outcome Quality Initiative (KDOQI) guidelines for routine assessment of calculated iron parameters to guide IV iron supplementation strategies [2,7].

Limitation(s)

Being a retrospective study, it was limited by available records. Inflammatory markers like C-reactive protein or Ferritin were not

uniformly available. In this study, the EPO dosing and iron therapy was not analysed.

CONCLUSION(S)

Based on the outcome of the study, it is implied that the iron i.v. supplementation is as important as diabetic and anti-hypertensive drugs. Biochemically, it is recommended to estimate all iron profile parameters post dialysis to implement precision medicine. Especially the calculated parameters; TSAT and UIBC will give a better insight regarding the physiological mechanism of iron storage and transport without molecular study of the same.

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